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Review Article

Classification, pathophysiology and principle of therapy of shock

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ABSTRACT

Shock is a clinical syndrome that results from lack of oxygen utilization or supply to vital organs resulting in hypoxia. Shock is associated with significant morbidity and mortality as if not treated timely can lead to multiple organ failure and death. The mortality rate of shock ranges from 20% to 50%. Shock has several pathophysiologies including intracardiac etiologies such as myocardial infarction, myopathy, or severe arrhythmia which produce altered maturation and cause heart failure while loss of internal or external fluid due to trauma or bleeding often results in hypovolemia. Also, obstruction by extracardiac causes and activation of inflammatory cascade can induce shock. The purpose of this research is to review the available information about the classification, pathophysiology and principle of therapy of shock. The major classification of shock includes cardiogenic, hypovolemic, obstructive and distributive shock. Distributive shock is further divided into three subclasses of septic, anaphylactic and neurogenic shock. Haemorrhagic shock is also the sub-type of hypovolemic shock. Each class of shock requires a specific treatment and timely management to prevent any further complications. Constant examination, resuscitation and re-evaluation are important in therapy of shock. To treat hypotension and to increase cardiac output, vasopressor drugs and inotropic adrenergic drugs continue to be the most widely used therapies. Endogenous catecholamines such as epinephrine, norepinephrine, and dopamine among other vasopressors have been shown to be effective in treating various types of shocks that are controlled as part of treatment for shock. Early diagnosis and prompt treatment can help in prevention of complications of shock also enhancing the recovery of patients.

Keywords: Shock, Class, Therapy, Mortality

INTRODUCTION

The clinical state that results from abrupt circulatory failure that is complex and life-threatening is known as shock. When the circulation is unable to provide enough oxygen and nutrients to cells and tissues, shock develops as a pathological condition. If resultant hypoxia, tissue hypoperfusion, and cellular dysfunction are not treated promptly and appropriately, it may result in multi-organ failure and death.[1] Clinical outcomes have gradually improved as a result of improved in-hospital systems of care that include efficient resuscitation and supportive measures, prompt antibiotic treatment, and cardiac reperfusion therapy. Nevertheless, shock continues to be a clinical condition with a documented short-term death rate ranging from 20-50%, despite these advancements.[2]

Reduced oxygen delivery, increased oxygen consumption, or insufficient oxygen utilization that results in hypoxia in cells and tissues are characteristics of shock. The most prevalent symptom of this potentially fatal cardiovascular disease is hypotension which refers to systolic blood pressure less than 90 mmHg or mean arterial pressure less than 65 mmHg. Without prompt treatment, shock, which is the culmination of a long range of etiologies, could be lethal. Distributive, hypovolemic, cardiogenic, and obstructive shock fall mostly into the four basic categories of shock.[3] The first step in an assessment is to determine whether critical measures like intubation, mechanical breathing, or vascular access are necessary. Imaging and laboratory testing should be started right once, as needed, especially to check the amount of blood lactate. It is necessary to assess the intravascular volume status of shock patients in order to classify their condition and guide therapy choices and options. The likelihood that a patient will survive this potentially fatal disease can be improved by adhering to evidence-based treatment of the specific causes of shock.[4]

Shock results from one or more of the following four pathophysiological processes. While intracardiac etiologies such as myocardial infarction, myopathy, or a severe arrhythmia produce altered contractility and cardiogenic failure, internal or external fluid loss such as through trauma/ gastrointestinal haemorrhage frequently results in hypovolemia. Obstruction is brought on by extracardiac causes of cardiac pump failure, such as pulmonary embolism and tension pneumothorax. Distributive effects of inflammatory substances, such as sepsis, anaphylaxis, poisoning, or other vasodilation effects, frequently cause vascular permeability and loss of vascular tone, which result in distributive shock.[5] The purpose of this research is to review the available information about the classification, pathophysiology and principle of therapy of shock.

LITERATURE SEARCH

This study is based on a comprehensive literature search conducted on June 24, 2022, in the Medline and Cochrane databases, utilizing the medical topic headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any possible research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed the information about the classification, pathophysiology and principle of therapy of shock. There no restrictions on date, language, participant age, or type of publication.

DISCUSSION

Shock is a systemic condition characterized by signs and symptoms that reflect the response of various organs to a state of hypoperfusion for their cells' basic metabolic needs. Shock is an acute or hyperacute physiological disturbance. Shock can be classified into general categories of cardiogenic, haemorrhagic, inflammatory, septic and toxic shock. Because of its many etiologies and the wide range of available treatments, shock can occasionally be difficult to adequately classify and is frequently challenging to cure. Knowing the kind of shock is essential for effective therapy since treatments that are beneficial for one form of shock may be harmful for another. Additionally, for best results, it is needed to have a solid grasp of the physiology of the various types of shock as well as the pharmacology of shock therapy. From their first stage of compensated physiology through a stage of decompensation, shock syndromes' clinical features are detailed. When treatment is delayed or is insufficient, subclinical shock persists despite changes in the macrohaemodynamic variables and, if aggressively handled, progresses to a second wave of physiological deterioration. Drug-resistant hypotension is a defining feature of irreversible shock, which results from a direct hit or from insufficient or delayed therapy.[6]

Classification of shock

Four major classes of shock briefly described. Synoptic view of 4 major types of shock shows in Figure 1.[7]

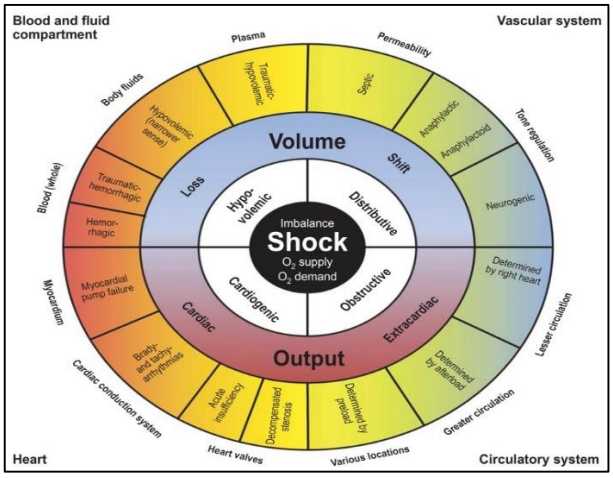

Figure 1: Synoptic view of four major types of shock.[7]

Cardiogenic shock

Cardiogenic shock is a physiological condition in which the heart's ability to circulate blood throughout the body is insufficient. Tissue hypoperfusion can soon result in organ failure and patient death if cardiogenic shock is not promptly diagnosed and treated. An electrocardiography, chest radiograph, laboratory tests, and bedside echocardiography should all be performed on individuals with suspected cardiogenic shock. The goals of the initial resuscitation are to improve tissue perfusion and cardiac output. Patients with cardiogenic shock who do not respond to pharmacological therapy should get mechanical circulatory support. These individuals should ultimately receive urgent reperfusion therapy via coronary artery bypass grafting or percutaneous coronary intervention.[8]

Hypovolemic shock

Inadequate organ perfusion brought on by abrupt intravascular volume loss is known as hypovolemic shock. The end result is a severe drop in ventricular preload, decreased macro- and microcirculation, adverse effects on tissue metabolism, and the induction of an inflammatory response. Haemorrhagic shock, traumatic haemorrhagic shock, hypovolemic shock in the stricter definition, arising from a catastrophic drop in circulating plasma volume without acute haemorrhage, and traumatic hypovolemic shock are the several subtypes of hypovolemic shock.[7] Haemorrhagic shock results from substantial blood loss results in insufficient oxygen supply to cells. It can cause death immediately if haemorrhage is left untreated. The causes of shock include trauma, maternal haemorrhage, gastrointestinal haemorrhage, perioperative haemorrhage, and rupture of an aneurysm.[9]

Obstructive shock

Clinical signs of shock, including confusion, concentration problems, oliguria, hypotension, and tachycardia, are brought on by a sudden restriction of blood flow in central veins of the systemic or pulmonary circulation. Intra-vascular occlusion causes an immediate rise in rt ventricular afterload in case of acute pulmonary embolism. An increase in extravascular pressure leads to an obstruction of blood arteries supplying the heart in a tension pneumothorax. From a hemodynamic perspective, cardiac deterioration immediately follows circulatory shock brought on by blockage; however, etiological and therapeutic choices demand separating cardiac from non-cardiac obstructive causes. All forms of obstructive shock share this potentially fatal condition's high dynamics as a defining characteristic.[10]

Distributive shock

The most common type of shock is distributive shock, which is characterized by a state of relative hypovolemia brought on by a pathological redistribution of the absolute intravascular volume. Either a loss of control over vascular tone, resulting in a change in intravascular volume within the circulatory system, or a disordered permeability of the vascular system, resulting in a shift in intravascular volume into the interstitium, are the likely causes. Septic, anaphylactic or anaphylactoid, and neurogenic shock are the three subtypes.[7]

Septic shock

Significant causes of mortality for hospitalized patients include sepsis and septic shock. The sepsis state results from an unbalanced host reaction to infection, which inflames almost every organ system. To lessen organ system damage and mortality, sepsis must be identified early and treated appropriately with antibiotics, fluids, and vasopressors.[11]

Anaphylactic shock

Following contact with the trigger, a number of substances are released that affect vascular permeability, smooth muscle tone of blood vessels, and the tone of the bronchi, as well as the activation of the systemic inflammatory cascade. Anaphylactic symptoms and anaphylactic shock are serious, rapidly developing, and potentially fatal systemic reactions. A substantial release of biologically active mediators from mast cells and basophils can accompany an immunoglobulin E-mediated immune response, according to the pathophysiology of the condition. The mastocyte and basophil are not allergenic if it degranulates directly through an immunoglobulin E-free pathway. Anaphylaxis is diagnosed using clinical criteria, keeping in mind the urgency of starting treatment for a condition that poses a serious risk to one's life. There is no contraindication for using adrenaline as the first-line treatment for anaphylaxis. In order for the patient to acquire hypotension, venous intake must be provided as soon as possible.[12]

Neurogenic shock

Especially when localized at the cervical level, the neurogenic shock is a frequent side effect of spinal cord damage. The neurogenic shock is a result of injury to the sympathetic nervous system and is characterized by vasoplegia, hypotension and bradycardia. Tetraplegia, with or without respiratory failure, is a common feature of the clinical presentation. Early intervention tries to reduce the likelihood of secondary spinal cord lesions brought on by ischemic systemic damage. Medical care involves using a systematic technique to immobilize the spine and stabilize essential functions. Imaging procedures, further neuro-resuscitation techniques, and coordinated surgical evaluation and treatment of any underlying injuries are all the included in the hospital care.[13]

Pathophysiology of shock

A series of physiological and biochemical changes brought on by tissue hypoxia at the cellular level can lead to acidosis, a reduction in local blood flow, and additional tissue hypoxia. There is a decline in cardiac output and a reduction in oxygen transport in hypovolemic, obstructive, and cardiogenic shock. Reduced peripheral vascular resistance and irregular oxygen extraction characterize distributive shock. From the earliest, reversible stages of excitement to the most severe ones, which result in multiorgan failure and death, there is a spectrum of physiologic changes associated with shock. Shock often goes through the following three stages: pre-shock, which is also known as compensated shock, is characterized by compensatory mechanisms that are used to offset the reduction in tissue perfusion. These include tachycardia, peripheral vasoconstriction, and changes in systemic blood pressure, shock-as the pre-shock stage progresses and the compensatory mechanisms fail, the majority of the characteristic signs and symptoms of shock manifest, this is because early organ dysfunction results from the evolution of the pre-shock stage and end-organ dysfunction is the last stage, which results in death, irreversible organ malfunction, and multiorgan failure.[14]

Therapeutic approach for shock

Shock is a medical emergency situation which calls for constant examination, resuscitation, and re-evaluation at the bedside. The initial bedside examination enables the clinician to ascertain whether the patient displays a clinical picture that is consistent with cardiogenic, vasodilatory, or hypovolemic shock. The prompt initial resuscitation recommended by the primary survey typically include intubation, ventilation, and volume support. Vasoactive therapy, which includes inotropic support for cardiogenic shock and pressor therapy for vasodilatory shock, is initiated once the patient has been adequately volume resuscitated. The secondary survey is essential for establishing early definite therapy and useful in identifying the shock's underlying cause. There is a hemodynamic component to early shock, which is frequently quickly reversible. Multiple-system organ failure and mortality are caused by an inflammatory component that is present in septic shock as well as chronic shock from any cause. Early detection of shock and a quick pace of hemodynamic resuscitation to stop or reduce the inflammatory component are essential to treating shock successfully.[15]

Fluids and vasoactive substances are linked in symptomatic therapy. To treat hypotension and to boost cardiac output, vasopressor and inotropic adrenergic drugs continue to be the most often utilized treatments. Although the haemodynamic and metabolic profiles of various drugs vary, the impact of these variations on the final result has long been disputed. Randomized studies conducted recently have shed some insight on this problem. The most in-depth research has been done on dopamine and norepinephrine. The use of dopamine, which was linked to tachycardia, an increase in arrhythmic events, and may also be related with an increased risk of death, particularly in the subgroup of patients with cardiogenic shock, was the subject of serious concerns raised by these studies. Epinephrine's role is not clearly established, however it is known to cause tachycardia, an increased risk of arrhythmic events, and undesirable metabolic effects.[16] Due to the numerous clinical manifestations of cardiogenic shock, septic shock, and hypovolemic shock as well as the limitations of available modern therapeutic options, managing shock patients is highly difficult. Endogenous catecholamines such as epinephrine, norepinephrine, and dopamine and other vasopressors that have shown effective in treating the various forms of shock are administered as part of the treatment of shock. Dobutamine, isoproterenol, phenylephrine, and milrinone have been the mainstays of shock therapy for many years in addition to the endogenous catecholamines. Experimental research has recently indicated that newer medications, including vasopressin, selepressin, calcium-sensitizing drugs like levosimendan, cardiac-specific myosin activators like omecamtiv mecarbil, istaroxime, and natriuretic peptides like nesiritide, may improve shock therapy, particularly when shock exhibits a more complex clinical picture than usual. It has yet to be demonstrated whether they can enhance clinical outcomes.[6] In recent times there is lack of research on the topic of shock also more clinical trials and research studies in future can aid in developing better prevention and treatment guidelines for therapy of shock also will add to the literature.

CONCLUSION

Shock is a significant medical condition associated with high death and morbidity rates. Distributive, cardiogenic, hypovolemic and obstructive shock are main classes of shock. Early diagnosis and management are essential because they can stop progression of condition to reversible shock, multiorgan failure, and death.

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